A This is PLT 12. This is GRN 12.
Q Yes, I got the wrong thing marked. We will just leave it. I will have to find that document and come back to it. It is marked as an exhibit and we will keep it as an exhibit. And so it will go in that pile.
Let me hand you 104. 104 is your questionnaire results for GRN 13; is that right?
(Defendants' Exhibit 104 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 13 is one of the samples that you sent to Axys labs for dioxin analysis; correct?
A Yes.
Q How old is GRN 13?
A 59.
Q White female; correct?
A Yes.
Q 5'2", 125 pounds?
A Yes.
Q Nonsmoker; correct?
A Correct.
Q 5'2", 125 pounds, is that obese in your view?
A No.
Q Deposition Exhibit 105 is your questionnaire

results for GRN 10; is that right?
(Defendants' Exhibit 105 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 10 is one of the samples that you sent to Axys Laboratories for dioxin analysis?
A Yes.
Q How hold is GRN 10?
A 41.
Q White male; correct?
A Correct.
Q Six-foot tall, 214 pounds; correct?
A Correct.
Q Is that obese?
A No.
Q Began smoking at age 15; correct?
A Yes.
Q And the most he has ever smoked is 30 cigarettes a day. So a pack and a half; right?
A Yes.
Q Is that heavy smoking?
A Yes.
Q Deposition Exhibit 106 is your questionnaire results for GRN 11; correct?

(Defendants' Exhibit 106 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 11 is one of the samples that you sent to Axys Laboratories for dioxin analysis; correct?
A Yes.
Q How old is GRN 11?
A 38.
Q White female; correct?
A Yes, white female.
Q 5'3", 228 pounds?
A That's what it says.
Q Is that obese?
A Yes.
Q Nonsmoker; correct?
A Correct.
Q Deposition Exhibit 107 is your results for GRN 14; correct?
(Defendants' Exhibit 107 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 14 is one of the blood samples you sent to Axys Laboratories for dioxin analysis; correct?

A Yes.
Q How old is GRN 14?
A 20.
Q White female; correct?
A Yes.
Q 5'7", 225 pounds. Do you see that?
A Yes.
Q Is that obese?
A Yes.
Q Nonsmoker; correct?
A Yes.
Q Deposition Exhibit 108 is your questionnaire results for GRN 06; correct?
(Defendants' Exhibit 108 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 06 is one of the blood samples you sent to Axys labs for dioxin analysis; correct?
A Yes.
Q How old is GRN 06?
A 38.
Q White female; correct?
A Yes.
Q 5'5", 200 pounds; is that right?

A That's right.
Q Is that obese?
A Yes.
Q Smoked since she was 15; is that right?
A That's right.
Q Pack a day?
A Wait a minute. 15 cigarettes a day?
Q It says, "15 years old."
A Oh, 20, yes, a pack a day.
Q In all of these questionnaires, you asked, what is the most that you ever smoked. There is no real way to know what their current level of smoking is; is there?
A No, not from that questionnaire. You have to do more inquiry.
Q Deposition Exhibit 109 is your sample results -- I'm sorry. Is your questionnaire results from GRN 08; correct?
(Defendants' Exhibit 109 was marked for identification by the court reporter.)
THE WITNESS: That's right.
BY MR. HOPP:
Q And GRN 08 is one of your samples that you sent to Axys Laboratories for dioxin analysis; right?
A Yup.

Q How hold is GRN 08?
A 72.
Q White female; correct?
A Yes.
Q 5'3", 165 pounds?
A Yes.
Q Is that obese?
A Probably just at the borderline.
Q Nonsmoker; correct?
A Yes.
Q Deposition Exhibit 110 is the questionnaire results for GRN 07; correct?
(Defendants' Exhibit 110 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 07 is one of the samples that you sent to Axys labs for dioxin analysis; correct?
A Yes.
Q How old is GRN 07?
A 35.
Q White male; correct?
A That's correct.
Q 5'10", 225 pounds; right?
A Yes.

Q Nonsmoker?
A Correct.
Q Is 5'10", 225 pounds considered obese in your view?
A No.
Q Deposition Exhibit No. 111 is your questionnaire responses for GRN 05; is that right?
(Defendants' Exhibit 111 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 05 is one of the samples you sent to Axys labs for dioxin analysis; is that right?
A Yes.
Q How old is GRN 05?
A 59.
Q White male; correct?
A Yes.
Q 5'9", but we -- but we don't see the weight there?
A No weight was put on the questionnaire. Correct.
Q Is that -- is that rare? I mean, I've never seen someone to refuse to answer their weight question or omit to put their weight.

A Just an oversight. Someone forgot to fill it in.
Q Nonsmoker; correct?
A Correct.
Q Almost done.
Deposition Exhibit 112 is your questionnaire responses for GRN 09; is that right?
(Defendants' Exhibit 112 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 09 was one of the samples that you sent to Axys Laboratories for dioxin sampling; is that right?
A Yes.
Q How old is GRN 09?
A 47.
Q And a white female; correct?
A Yes.
Q 5'6", 150; correct?
A Yes.
Q And is that obese?
A No.
Q Nonsmoker; correct?
A Yes.
Q Deposition Exhibit 112 is your sample

results -- I'm sorry. Is your questionnaire responses for GRN 03; correct?
(Defendants' Exhibit 113 was marked for identification by the court reporter.)
THE WITNESS: Yes.
MR. PRUDHOMME: This would be 113; wouldn't it? You said, "112."
MR. HOPP: It is 113.
Q Your deposition Exhibit 113 is questionnaire response GRN 03; correct?
A Yes.
Q And GRN 03 is one of the samples that you sent to Axys Laboratories for dioxin analysis; correct?
A Right.
Q How old is GRN 03?
A 40 -- this is interesting. She filled this thing out in 2004 and they were just about almost -- they were 43.
Q 43. 5'2", 160; right?
A Yup.
Q White female; correct?
A Yes.
Q Nonsmoker; is that right?
A Correct.
Q Is 5'2", 160 obese in your view?

A Borderline.
Q 114 is your questionnaire results for GRN 02; is that right?
(Defendants' Exhibit 114 was marked for identification by the court reporter.)
THE WITNESS: That's right.
BY MR. HOPP:
Q And GRN 02 is one of your samples that you sent to Axys labs for dioxin analysis?
A Yes.
Q How old is GRN 02?
A 53.
Q White female; correct?
A White female; correct.
Q 5'7", 159; right?
A Yes.
Q Is that obese?
A No.
Q Nonsmoker; correct?
A Correct.
Q 115 is your questionnaire responses for GRN 01; correct?
(Defendants' Exhibit 115 was marked for identification by the court reporter.)
THE WITNESS: Yes.

BY MR. HOPP:
Q And GRN 01 is one of the samples that you sent to Axys labs for dioxin analysis; correct?
A Yes.
Q How old is GRN 01?
A 19.
Q White male; correct?
A Right.
Q Six-foot, 162 pounds; is that right?
A Yes.
Q And that is not obese; is it?
A No.
Q Nonsmoker; correct?
A Correct.
Q 116 is your questionnaire responses for GRN 04; is that correct?
(Defendants' Exhibit 116 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And GRN 04 is one of the samples that you sent to Axys labs for dioxin analysis; correct?
A Yeah. I am just looking for the sample sheet here.
Q It's the last sample preparation record before

the pooled samples, at least in my copy of documents.
A Here it is. Okay.
Q Okay. Now, GRN 04 is one of the samples that you sent to Axys labs for dioxin analysis; correct?
A Yes.
Q How old is GRN 04?
A 24.
Q White female; correct?
A Yes.
Q 5'4", 177 pounds; is that right?
A That's right.
Q Is that obese?
A Yes.
Q Nonsmoker; correct?
A Yes.
Q Now, the deposition Exhibit 13 contains all of the sample preparations for all of the samples you sent to Axys labs for dioxin analysis; is that right? There aren't any missing dioxin samples; are there?
A Well, let's see, have we done GRN 03, 02, 01? Yes, we did.
Q I think we did.
A All right. Yes, I guess that's it. I did not count them, but I will take your word for it.
Q What would be representative of the Axys

report; that is, everything that went to Axys for the purpose of dioxin analysis; right?
A Yes, I think so. And we --
Q And as we discussed, that was the origin of the four pooled samples; right?
A Right.
Q Is there any data that -- strike that.
Under whose instructions, did Axys pool the 4 -- 40 individual samples into four composite samples?
A Me.
Q And was that based on cost?
A Yes, as I have testified. In order to see whether the group as a whole had at high values compared to -- comparison group and compared to other published norms, we wanted to be more economical. And then, ultimately, if the values were high in the exposed group especially, we would have the opportunity to go back and do individual samples.
Q Do the individual serum or whole blood samples that are described in deposition Exhibit 13 still exist in the freezer up in Axys labs?
A I don't know. I haven't checked. I think we asked them to hold onto them until we analyzed the results.
As soon as we analyzed the results and realized

that there wasn't going to be anything -- anything of importance to the particular case that was involved here, we let them throw them away.
Q Now, I apologize if we covered this, but just for context, blood samples containing dioxin have a long shelf life if they are properly handled; right?
A Yes. If you keep them frozen, you can analyze them later.
Q Dr. Schecter has, in fact, gone back recent years, have analyzed samples taken in the 1970's?
A Yes, we talked about that this morning.
Q So those were frozen and then he got samples of the frozen blood of 25 years ago or 30 years ago and looked at those; is that right?
A That's right.
Q Would there be any way to tell now what the individual results were for the -- strike that.
Would there be any way to tell now what the individual dioxin results would have been for the individual samples collected and sent to Axys labs?
A No. No, you would have to do the individual analysis to answer that question.
Q Let's talk about the control values for PAHs.
Now, you testified a couple of times that the exposed population for the PAH results was comprised of

the same group of people that you used for your dioxin analysis; is that right?
A Well, I think there is -- yeah.
Q There is a few, a couple didn't show up?
A There are some people that didn't get into both lists, but that was our plan.
Q Now, let's look at Page 53 -- 52 and 53 of your report in this case. I think -- you got it?
A Okay.
Q Now, I want to direct your attention to Page 53, the comparison samples. Do you see that?
A Yes.
Q They have seven comparison samples; is that correct?
A Yes.
Q And looking at this or from memory, can you tell me where these seven comparison samples originated?
A Well, from our Florida work. I don't know which ones this relates. It does not represent -- I think this is probably Jerome or Homosassa, one of the two.
Q That is what I want to try to tie down.
Deposition Exhibit 68, which is your PAH results, I tried to track them back against the comparisons on Page 53 of 305 of your expert report and

it appears to me that the numbers track with the Jerome plaintiffs rather than the Homosassa controls.
Can you verify or dispute that?
A No, I think that's right.
Q All right. So you used Jerome as the comparison population for Grenada; correct?
A Correct.
Q Why?
A I don't recall at this moment why I did that.
Q Do you know why you didn't use both Jerome and Homosassa?
A I think there was some question about the Homosassa results that was raised. I don't recall the details. But as you see, there are two results there and an A and a B. And there was some problem with the runs that caused them to be more wide than they ordinarily should be when you run duplicates like that.
Q Okay.
A So I think that is why we rejected them because they should have been closer -- the duplicate should have been closer.
Q Just so I understand that answer, you are looking at deposition Exhibit 68?
A Yes.
Q Let's take James Mitchell, Jr., for example,

the two runs, one of the values is .75 and the other is 4.15; is that right?
A Correct.
Q When you say "two runs," is that the same sample protocol run on the same blood coming up with two different numbers?
A Yes, I think that is what they did. They do that ordinarily. But if they are close to each other, they don't question it.
If there is a scatter, then they report both and you can decide whether or not you want to throw out the results completely.
Q Okay. I am trying to understand this.
The top of the column, again, looking at Page 68, the top of the column with the results in it, it says, "Ave of 2 Expts." Do you see that?
A No. Where are you looking?
Q Deposition Exhibit 68, which is the spreadsheet from the lab.
A Yeah.
Q And then it says, "Ave of 2 Expts," E-X-P-T-S. What does that mean?
A Two experiments, I think that is what it means. But when they are -- when there is disparity, then they report both.

I don't know what the cutoff is, but the Homosassa samples, what happened is that those people took a bus down to Miami and had the blood samples pulled there. And there was some problem with the draw and with the handling of those specimens.
All of them had some kind of a problem with them. I don't remember the details, that is why we didn't include them as the exposure measurements.
Q Well, just, again, so we are clear on Homosassa. The Homosassa results on deposition Exhibit 68, you don't have an average. You have both results reported?
A I already indicated, that is because they were -- if they are close together, they just average them. But if they are wide apart, they don't.
And there was some question about this whole batch. And, you know, it was unfortunate, but, you know, that is what the data showed. So we just decided not to use them as a comparison group because they were inconsistent.
Q Do you have the same problem with Simmons A and Simmons B?
A Yes, same problems. Also was too far apart.
Q Now, do you know what the -- strike that.
For the Jerome plaintiffs, who we see

represented without names on the front page of deposition Exhibit 68?
A Right.
Q You have single value because that means it is an average; right?
A That's right.
Q Are the individual values that make up the average reported anywhere that you are aware of?
A No. As I stated, when they are consistent, they just have the one number.
Q The lab would have that somewhere, though; right?
A I would presume they would, yes.
Q How was the Jerome -- strike that.
And, again, I apologize if we covered this.
You have 50 people in Jerome. You have seven people who were tested. I believe you testified earlier that the cost was a factor?
A No, it was availability.
Q How come you got only seven folks from Jerome?
A Well, I think the real issue was that there was only seven people that were still being exposed --
Q Okay.
A -- theoretically.
In other words, that exposure was to creosote

in the water and they were still living on the property where the contamination occurred; but they were drinking bottled water; and they were using filters on their bathing water.
We thought they might be still exposed. So we did this sampling. The rest of the plaintiffs were living elsewhere and were not exposed. And PAH adducts are only useful on an ongoing acute basis.
Q Now, in Jerome, PAHs were identified in the water quite some time ago; 1990 or so or before?
A Yes. Yes, this particular group of people were exposed probably dating back to the '50s, '60s, '80 -- late '80, early '90.
It was discovered they went on bottled water. In fact, when they checked the water in 2003, it was very little detected, which is totally consistent with what is seen here. In other words, they didn't really have any exposure.
Q So they were -- at least from 1990 forward, these people were drinking bottled water?
A Yes. I don't know the exact year, but some time quite a ways back, they were put on bottled water.
Q And someone who would have moved into the neighborhood then, after the bottled water was provided, would not have had that exposure pathway; correct?

A That's right.
Q Now, again, I am just trying to understand your prior answer. You said that you thought there might be some ongoing exposure, then, to the Jerome individuals we see identified in deposition Exhibit 68?
A Yes.
Q If it wasn't the water, what was it?
A Skin contact, maybe from the bathing. Maybe from the soil. We just wanted to check.
Q So they did at least live in the area that was served by water that at one point --
A They still did. That's right. And I think they were the only seven. That is why we ended up with only seven.
Q How was -- let me back up.
You testified a minute ago that there was in -- strike that.
You testified earlier that the Homosassa folks were bussed from Homosassa, which is north of Tampa on the Golf Coast?
A Yes, it is a long bus ride. That was the only place that we could find that could do the sample prep.
Q Down in Miami?
A Miami.
Q Where is Jerome in relation to Miami?

A It is close to Naples. Jerome is not even an incorporated city. It is just a wide spot in the road with a name.
Q Were the Jerome plaintiffs also bussed to Miami for a blood draw?
A I think so. I am trying to remember how we got those blood -- actually, I think they were drawn out in Jerome or in that area and then taken quickly by messenger or by courier; but Homosassa was so far away, that that wouldn't work. So they ended up traveling down to get it drawn.
Q Do you know how, if at all, the long bus drive would have affected the level of PAH specific in DNA adducts in the blood of the Homosassa people?
A No, I don't. I mean, I've never seen any data on that question.
Q And do you know if it was the bus drive that was a problem or rather the lab issue?
A No. The sample, the handling of the lab that caused the problem.
Q The samples are actually packed in --
A Not the laboratory in England, but the laboratory who drew the samples in Miami.
Q That the blood was drawn in Miami, packed in dry ice, and shipped to --

A No, it had to be processed to what they call the ficol method. F-I-C-O-L.
This was when they concentrate the white cells from the samples, the blood they collect; and they spin it down. And they separate the white cells and the red cells from the serum. And that is when they test the white cells.
Q So there was some mishandling of the ficol procedure that resulted in the wide variations you believe?
A Yes.
Q It is your assumption that you did not have the same disparity in the sample results in the Jerome people because Jerome was reported as a single value?
A Yes, that's Mirex.
Q Do you have handy on your computer your copy of the spreadsheet results that you got from Dr. Phillips in England?
A No, that is back on the server back at the office. Don't have that here.
Q Let's see if you can answer these questions. I am going to try to go through the Jerome plaintiffs and match them up -- try to match them up.
The Jerome spreadsheet that I got on a disk from plaintiff's counsel had two tabs to it. Let me see

if I can find the second tab.
The first tab had the Jerome sample results without names. The second one had the Jerome names.
Here we go. This can do it for us.
MR. PRUDHOMME: I have a call with a federal judge.
MR. HOPP: Let me clean up and -- is that all right, Doctor? Take a break for a few moments?
THE WITNESS: Sure.
(Brief recess.)
(Defendants' Exhibit 117 was marked for identification by the court reporter.)
MR. HOPP: Let's go back on.
Q Dr. Dalhgren, I am handing you what we have marked as deposition Exhibit No. 117 and that is my only copy. So I can't let you keep a copy of it.
That is a printout of the second tab of the spreadsheet, as we see identified as Exhibit -- deposition Exhibit 68.
Do you recognize the names on Exhibit 117 as the Jerome plaintiffs?
A No, I don't have any independent recollection of the names.
Q Is there any way using deposition Exhibit 68 -- let me show you, not to confuse things.

Let me hand you deposition Exhibit 118.
(Defendants' Exhibit 118 was marked for identification by the court reporter.)
BY MR. HOPP:
Q Deposition Exhibit 118 is a questionnaire from one of the Jerome plaintiffs, and for confidentiality reasons, the name has been blocked out.
Is there any way, using deposition Exhibit 68 or deposition Exhibit 117, you can tell me what the name of the plaintiff is whose questionnaire results are represented in deposition Exhibit 118?
A No, I don't --
Q All right. Do you have any reason to disagree with the statement that deposition Exhibit 118 represents the questionnaire results for Darcy Kidder, K-I-D-D-E-R?
A I don't know.
Q Do you know if Darcy Kidder was a medical monitoring plaintiff?
A No, I don't know.
Q Do you know if she lived with Lee Kidder?
A Well, they have the same last name. This is bad, but I don't have any independent recollection about that.
Q Do you know if she moved into Jerome after

1990?
A No, I don't. I have no way of knowing that without more -- finding more information.
There should be a residence history here. The one that you handed me, the residence in Jerome was from '71 to '73, and then in Copeland, down a road apiece, '73 to '90.
Q And then back to Jerome. Do we know that?
A They listed in their address from Jerome from '90 forward for 14 years. So that would have probably been the sequence of events.
Q Deposition Exhibit 18 is a 32-year-old white female; is that right?
A Yes.
Q I'm sorry, 118. She is 5' tall, weight, 140?
A Yes.
Q Nonsmoker; right?
A Yes.
Q Deposition Exhibit No. 119 is another Jerome plaintiff. Do you see that?
(Defendants' Exhibit 119 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q Is there any way that you can tell me the name

of this person?
A No.
Q And how old is the person whose questionnaire results are represented in deposition Exhibit 119?
A 11.
Q And what I am showing you -- what I have showed you is deposition Exhibits 118 and 119, and then on through the next seven or eight exhibits, are the questionnaire results that your office produced that relate to the Jerome plaintiffs.
Deposition Exhibit 68 shows results for the Jerome plaintiffs and I think you testified earlier that there was only a group of seven or nine Jerome plaintiffs that showed up for testing; is that right?
A That's right.
Q So if I got a questionnaire from you that says Jerome on it, that is one of the people whose results -- I'm sorry -- whose blood was sent to England for PAH analysis; correct?
A That's correct.
Q An 11-year-old who is represented in deposition Exhibit 119, is 5'2", 125 pounds; is that right?
A Yes.
Q Is that obese, in your view?
A Well, you know, I -- 5'2" and 125, a little

chubby, maybe.
Q Is it unusual for an 11-year-old girl to be chubby?
A No.
Q Next document is 120.
(Defendants' Exhibit 120 was marked for identification by the court reporter.)
BY MR. HOPP:
Q Deposition Exhibit 120 is another set of questionnaire results for a Jerome plaintiff. How old is the person represented in Deposition Exhibit 120?
A 26.
Q And this person gives an address as Copeland, C-O-P-E-L-A-N-D, Florida?
A That's correct.
Q Where is Copeland as a result to Jerome?
A About five miles down the road.
Q This person that we see in Exhibit 120 ever live in Jerome?
A Well, let's see. There should be some listing of address in here someplace. I think we have that as per the questionnaire, but I don't see it.
So, apparently, this particular questionnaire, which is different than the other one, doesn't have the residents history on it. So we don't know. We would

have to rely upon verbal history from this person.
Q Okay. Let's look at the questionnaire itself just for a moment. At the bottom of deposition Exhibit 120 it says, "Copyright Comprehensive Health Screening Services, Inc."
Do you see that?
A Yes.
Q And some of the questionnaires that we have been looking at, it has a copyright, James Dalhgren Medical?
A Right.
Q I assume James Dalhgren Medical is a company that you own?
A No, it is a sole proprietorship.
Q So that's you?
A That's just my practice.
Q Are you associated or have you ever been associated with a company called Comprehensive Health Screening Services, Inc.?
A Yes.
Q Can you describe your relationship with that company?
A Well, it's a corporation that we -- that I set up with a partner. 50 percent ownership for each of us. The other partner's name is Ray Warshaw. And he and I

worked on some of these screening projects together.
Q Did he work on the Jerome project?
A Yes, he did.
Q Do you still use the Comprehensive Health Screening Services health questionnaire?
A The last time we used it was about a year ago. We are not going to use it anymore.
Q Why is that?
A Because we have broken up our partnership.
Q So the work that you do that involves using a questionnaire from here on end, at least until things change, is going to be James Dalhgren Medical?
A Yes. One of the reasons -- this was an earlier questionnaire that we did. And, you know, it has no residence history, which is Ray Warshaw's responsibility. So anyway, that's part of the reason why we are not a partnership anymore.
Q Okay. Is there anything else that you find -- strike that.
Is there any other issues with deposition Exhibit 120 or Comprehensive Health Screening Services, Inc.'s survey forms which you changed -- let me ask a better question.
You are currently using the James Dalhgren Medical survey form; right?

A Right.
Q In the past, you used the Comprehensive Health Screening Services form; correct?
A Correct.
Q One of things that you testified is the things that you have now that you didn't have back then was the residence?
A Yes. We had it, but it was not attached to that particular questionnaire; but it should have been there because residence is an important issue.
Q Especially in Jerome because the issue was whether they were drinking the water?
A Or exposed to the water in any other way.
Q Is there any other problems that you found with the Comprehensive Health Screening Services' form which you corrected when you did your James Dalhgren Medical, Inc., survey?
A It is not that we corrected it, but we made sure that it was complete. We use the same questions.
For the most part, we will individualize questionnaires based upon certain things, specific things, meaning we add questions; but the basic questionnaire is the same.
Q All right.
A So, therefore, we can compare results as we go

forward.
Q And is there any way, based on the exhibits we got, particularly deposition Exhibit 68 and deposition Exhibit 117, that you can tell me who the person is whose results are represented in deposition Exhibit 120?
A No, I can't.
Q Do you remember that the 11-year-old in the Jerome case was named Laura Brown Sanders?
A No, I don't remember.
Q Looking at deposition Exhibit -- looking, again, at deposition Exhibit 120, I just want to know whether she was a smoker ever.
120 is a white female, 5'6", 140 pounds; correct?
A The smoking question should be here. We hope.
Q Here it is. I'm sorry. Page 3, nonsmoker; correct?
A Yes.
Q Deposition Exhibit 121 is your survey results for another Jerome plaintiff; correct?
(Defendants' Exhibit 121 was marked for identification by the court reporter.)
THE WITNESS: Yes. By the way, there was some residence history here on this one.

BY MR. HOPP:
Q I'm sorry. 120?
A 121, second page. She describes living in Jerome from age 20 to age 32, which is her current age.
Q Okay.
A Oh, no, his current age.
Q So 121 is a white male, 32 years old?
A Yes.
Q 6'2", 288 pounds; correct?
A That's correct.
Q Is that obese?
A Well, I guess, you would say he was obese. He is -- you know, there are people that -- well, yeah, I would say he is obese.
Q And he is a smoker; correct?
A No. It says, "No."
Q Well, he got cigars or something. He has got an X?
A There is an X next to the cigars. He accidentally answered yes and he crossed it out and answered it no.
Q Is there any way that you could tell whose -- who the subject of deposition Exhibit 121 is; the name of that person?
A No.

Q 122 is another set of questionnaire answers from Jerome; is that right?
(Defendants' Exhibit 122 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And what is the age of the person we see represented in deposition Exhibit 122?
A It seemed to be missing some pages with the age information. So I don't know what the age of this person is.
Q Well, again, I don't think I miscopied.
A Here it is. They weren't in the proper order. Page 1 is in the middle somehow. There are two parts to different Page 1.
Q She is 28, white female; right?
A Yes.
Q 5'7", 175 pounds?
A That's it. Um-hmm.
Q Is that obese?
A Borderline.
Q Nonsmoker; correct?
A Yes.
Q Lives in Copeland, Florida; is that right?
A Well, yeah. I think Copeland, by the way, is

actually the post office for Jerome.
Q Okay.
A So you can't -- Copeland is a little area a few miles down the road, but her actual address, I think, is Jerome.
Q And she listed her residence history as having lived in Jerome from age 1 to 27?
A And that's right. And she is 28. So maybe she moved away a year ago.
Q Deposition Exhibit 123, this is another Jerome plaintiff; correct?
(Defendants' Exhibit 123 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q This is a white male; right?
A Yes.
Q 33 years old, 5'11", 245 pounds; right?
A Yes.
Q Is that obese?
A Yeah, probably.
Q Now, this person gives an address of Everglades City and says that he was born in Fort Myers; is that right?
A That's right.

Q Can you tell, from this form, how long, if at all, this person lived in Jerome?
A Well, in the different type of residence questionnaires, it is near the back. Highway 29, Jerome. It doesn't say when to when.
Q Deposition Exhibit 124 is another Jerome plaintiff; right?
(Defendants' Exhibit 124 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q 45-year-old, white female; correct?
A Yes.
Q Five -- I'm sorry. 5'6 1/2", 180 pounds; correct?
A Yes.
Q Is that obese?
A Borderline.
Q Once again, gives the address of Copeland and states that she was born in Wilmington, Delaware; right?
A Yes.
Q Can you tell from this form how long, if at all, the person represented in deposition Exhibit 124 lived in Jerome, Florida? I think it is somewhere in the middle.

A 2000 to the present, lived in Jerome. So she was still living there.
Q Okay. That is after Jerome started to get bottled water; correct?
A Yes.
Q Deposition Exhibit 125, another Jerome plaintiff; correct?
(Defendants' Exhibit 125 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q 49 years old, 5'7", 185 pounds?
A Yes.
Q Nonsmoker; correct? I'm sorry. Former smoker? This is Page 3.
A Yes, he smoked from 18 to 35.
Q And his high was a pack and a half a day; correct?
A That's right.
Q On this form 125, you have a box on the top of the form that says, "CHSS staff only"; correct?
A Yes.
Q And there is an initial -- there is initialed in there, I think, "Verified: CAH"?
A Uh-huh.

Q Who is that, if you know?
A I don't know offhand.
Q When Comprehensive Health Screening Services, Inc., was in operation, did it operate out of your offices here in Santa Monica?
A Yes.
Q And would CAH had been someone on your staff at that time?
A Well, it could have been one of Mr. Warshaw's people or it could have been one of mine. I just recall those initials.
Q All right. Did Mr. Warshaw sometimes bring other folks with him to work on Comprehensive Health Screening Services, Inc., projects?
A He always did. He always brought several technicians from his shop.
Q And they would work under the heading of Comprehensive Health Screening Services?
A That's right.
Q So you don't know whether CAH was someone who worked for you or someone who worked for Mr. Warshaw?
A No, I don't.
Q Let's talk about Dr. Phillips. His name is David Phillips; is that right?
A Dr. Phillips is David Phillips, correct.

Q And he works for the section of Molecular Carcinogenesis Institute of Cancer in Sutton, England; is that right?
A That's right.
Q Would you have any objection to my taking a deposition of Dr. Phillips and asking him some questions about the work that was done on your behalf?
A No.
Q Would you have any objection to me contacting Axys Labs and setting up the deposition of their people to ask about the work done on your behalf?
A No.
Q When you contacted Dr. Phillips for the purpose of the work in this case, was there a work order or set of written -- was there written communication which summarized what you wanted him to do?
A Dr. Phillips?
Q Yes, Dr. Phillips.
A Well, I'm sure I sent him a letter. We communicated by telephone and by E-mail. Probably the communication was E-mail and I told him, you know, what we were doing and what we were looking for. And we communicated.
MR. HOPP: All right. If you still have copies of those communications, I am going to make a request

now on the record and ask Keith, hopefully, to coordinate that. I would like copies of those.
MR. PRUDHOMME: Just for the record, if you want to take Dr. Phillips' deposition, please coordinate through us.
MR. HOPP: Oh, that's fine. Same goes with Axys?
MR. PRUDHOMME: Right. Tell me again now.
MR. HOPP: Written communication with Dr. Phillips describing the nature of the project and, you know, basically his assignment.
Q The reason I ask, Dr. Dalhgren, is that I don't see a lab report from Dr. Phillips that is a report similar to what you have got from Axys and what you got from ERGO Laboratories setting --
A That is because Dr. Phillips is not a commercial lab. He does not have a regular reporting system. So he -- you know, he gave the results on a spreadsheet.
And, you know, it is not like commercial labs where they have a whole reporting system in place.
Q The spreadsheet is what you produced on a disk in which you produced in this case?
A Yes.
Q So what we see represented in deposition

Exhibit 68 is --
A Is pretty much what we got, yeah.
Q Returning for a moment to the Jerome plaintiffs, when I was asking questions about selecting them and how you found them; and how you were limited with who you could take samples from, you used the term "we" in describing the work that was done to identify and bring the Jerome plaintiffs for testing. Who is "we"?
A My staff.
Q Were the lawyers involved in contacting the Jerome plaintiffs for the purpose of getting them in for analysis?
A Well, the lawyers representing them in the lawsuit, yes.
Q And who were the lawyers representing them in the lawsuit?
A Don Russo.
Q Now, in selecting the controls for the purpose of your work in this case, did you make any attempt to match the controls to your exposed population, either when it came to the dioxin results or the PAH results?
A Well, I think I already testified that these were a convenience comparison group that we had studied. They were not specifically matched, no.

We -- the Jerome group turned out to be a group of people that had levels similar to what you would expect to see in the general population and significantly different than the group in Grenada.
But we didn't make any attempt to match them ahead of time. As I said, it was a convenience situation where we had this data and was able to, you know, utilize it.
Same is true with the Greenville case, as I have already testified. That was collected for reasons that I have stated and not specifically to be a controlled group for this population; but it turned out to be, you know, a convenience comparison group because of the similarities of exposure.
Similarity of background exposure, similar of size town and state. And, you know, they are in the southern part of the United States, in a small southern town. Not exactly the same but, you know, close. So it seemed like an appropriate comparison.
Q Is it standard procedure, when doing a comparison like this, to try to match cases and controls by age, gender, ethnicity, and body weight, and other factors?
A Yes. When you are doing an epidemiological study and you have funding to do so, that would be the

ideal, but in this case, I didn't have that.
All I had was the levels we measured in Grenada and I had to compare them to other -- in the case of adduct, we did it simultaneously, at the same time, which is the key point that Dr. Phillips makes is that if you are going to do the comparisons, you need to run the samples at the same time because there is variability in one to run.
You don't want to do one group on one run and then say, six months later, do a run on another group and then compare the two.
You want to run the two together because you want the conditions to be very, very similar or close as possible, so you are doing it all at once, treating everybody the same way. Phillips didn't know who the exposed or unexposed were. He did the samples as they came.
The same with the Greenville and the dioxin. We -- you know, there was no prejudging of that. We didn't, as I said, go out and say, here is the people we have in Grenada. We are going to find someone similar for controls. We just happened to have this data and I think it has been useful.
Now, I got to say, at that time, we also looked at other data that is available and I think it is, you

know, consistent with what we saw in Greenville is consistent with what is in the literature. And, you know, with all of the caveats that we asked earlier.
Q Are there reported values for -- let me back up.
What, precisely, was Dr. Phillips seeking to measure? We kind of danced around this, but I want to get a better definition of Dr. Phillips' duties. We called it PAH DNA adducts?
A Yes.
Q What was Dr. Phillips looking for in the blood that he was testing?
A Well, it's the P32 post-label technique and basically, what they do is they incubate the white blood cells with this P32 and the P32 reacts with the adducts incorporates -- is incorporated into DNA, PAH, adduct, and is tightly bound to that adduct. And this is, you know, something that has been learned over time, you know, that happens.
So now you got the adducts, the PAH, DNA, adducts that are now attached to a radioactive phosphorus molecule. You can then count the radioactivity and the radioactivity corresponds with the amount of PAH adducts that are present in that person's white blood cells. That's, basically, what they do.

Q And then you count up the number of radio --
A Radioactivity. They put the blood -- not the blood, but the prepared sample from the dissolved and shoot up white blood cells and they put it on the electrophoretic patterns going in two different directions and solvent systems.
Separate out the DNA adducts and the solubility system in the solvent. And then they take that filter paper and can't do radioactive counting on it, and you can see hot spots where there is a higher concentrate of the adducts in absence of a spotted -- so the intensity of the radioactivity tells you that it correlates with the concentration of the adducts that are present.
Q Is the process of counting the hot spots on the filter paper a manual process or done by a machine?
A Done by a machine. A Geiger counter, as I understand it. I am not an expert in this, but you have to ask Dr. Phillips the details.
In general, they put it on the paper -- the chromatographic paper that I stated and they put it in the machine and it is counted.
Now, I don't know whether they cut the paper in pieces or whether they scan it, but they count the radioactivity.
Q Somewhere there is a published standard for P32

A Nucleotide. What is it? Nucleotides.
Q What is a nucleotide?
A It a component of the DNA.
Q What is an adduct?
A It is in my report. I got a page where there is a picture of an adduct, basically, or a graphical representation of what an adduct is.
It is where the PAH molecule actually forms a chemical bond with the DNA and so it is the DNA, plus the PAH together. When the two are together, this is called an adduct.
Q So what you are looking for is the number of adducts per 10 to the minus 8th?
A No, 10 to the 8th.
Q I'm sorry?
A 10 to the 8th nucleotide.
Q Now, are their literature values, published literature values for levels of DNA adducts in blood?
A Yes, there are, but as I just indicated to you, there is a great deal of variability. So you can't really compare a number published by one group with a number published by another. It always has to be relative to something.
Now, they are close. I mean, they are not widely different, but you can't take a, you know .58 in

post-labels technique?
A Yes, this has been around for decades. Very well-established.
Q There is also a technique called Alyssa; is that right?
A Correct.
Q Do you know why Phillips used P32 post-labels as opposed to Alyssa?
A Historically, it has been more sensitive. With the Alyssa method, you frequently don't see anything because its detection limits are higher. Whereas, with the Post 32 labels technique, you almost always see some adducts even with the most minimally exposed people. So that is why I think he used it.
Q Now, looking, again, at deposition Exhibit 38, which is the printout of Dr. Phillips' results, we see numbers, just looking at -- Simmons A and Simmons B or the Jerome.
Let's just take the first Jerome number, which is the third one down on deposition Exhibit 68. It is .58. Do you see that?
A Yes.
Q .58 what?
A It is .58 adducts per 10 to the 8th nucleides.
Q Nucleides or nucleotide?

one and a 5 in another, and they may be the same because the conditions of the testing would be the same. So you really need simultaneous controls, usually, when you are doing this study.
You have the exposed and the controls and you run it simultaneously and compare the numbers. There are sort of rough ranges that everybody would -- if it was too far high or too far low, they would question it.
Because even the most heavily exposed coal oven workers don't go sky high, and even the most unexposed general population person doesn't go too far down on the, you know, the lower side. There is a range that you generally see.
Q But correct me if I am wrong, it appears what you are saying is in contrast to the dioxin literature where there are published background levels of dioxin in blood nationwide, if not worldwide, there doesn't seem to be a body of literature which gives you a good example of what a background level would be for DNA adduct; is that right?
A I think that is a fair statement, yes. Because if you look at the literature, you will see a lot of variability.
Q And if you look at the literature, often what you see is heavily exposed population. A lot of this

work has been done in coal oven workers and people who have high levels of PAH exposures?
A Yes, but it has been done in lots of backgrounds. Done in children living in busy roadways compared to children who live far away from the roadway.
It has been done on people who live in communities next to PAH generating activity and you can see clear differences in people close by and versus control, who is a little further away. So it has been used in exactly the same setting done here.
Q Each time it is used, it is comparing one group to another group and not to establish an overall background normal level; right?
A As I have stated, it is not -- the standardizations of the technique is not such that you can do that. They keep talking about it and hoping that eventually they will get it down to the point where it is done so exactly the same that you can compare one lab and one run with the other. But at this moment, it is still not possible to do that.
Q Going back to a standard epidemiological study where you have funding and time, would it be standard to match cases and controls for age?
A Yes.
Q Would it be standard to match ages and controls

for gender -- strike that.
Would it be standard to match cases and control for gender?
A Yes, and we did that in Columbus. We did a formal epidemiologic study where we went out and found a town that was very, very similar to the town that we were studying, and we matched the populations as close as we could.
It turned out that there were some differences that we had to adjust for in the statistics. Inasmuch effort that we made to match them, there was still some discrepancies and that always happens. And what you do is you adjust -- let's say, there is a slight difference in the case of Selma and Columbus, there was a slight difference in the height of the population and we had no idea what we were going to find.
There were poor blacks living in the town and they were otherwise matched very closely, but the -- their heights were different. It was a significant difference in height.
And we also found a difference in educational level, which we did not expect; but it wasn't great. And we adjusted the statistical analysis taking those variations, but you always try to match as close as you can.

Q And just to go through the -- through the list of variables, do you also try to match when you can for ethnicity?
A Yes.
Q Do you try to match as best you can for body weight?
A Yes. Although that usually requires that you check the people, examine the people or run the questionnaire or know that ahead of time.
When you go looking for a control group, you are not going to know how much they weigh. So if there is a difference, you have to adjust for it.
In the case of Columbus and Selma, we don't have to adjust for body weight. We selected only for comparison purposes only, the blacks, so there was no question of differences in races.
Q And, typically, did you try to match for smoking?
A Usually you have to adjust for smoking. And in most cases, if you have a descent sample size, the smoking prevalence would be the same, but if not, you would adjust for it.
There is no way, ahead of time, you are going to know what smoking habits are going to be. If you match, as we did demographic, similar social, economic

group, regional group, things like smoking level and income level somewhat tend to match fairly closely.
Q Describe for me -- strike that.
Again, the purpose of context and using Selma and Columbus as an exemplar, you have two different groups of people, it could be in the hundreds or the thousands. I think in Selma you had a few hundred and in Columbus you had over a thousand; is that right?
A Well, yes, we -- I am forgetting.
Q Let me find the article.
A The questionnaire survey involved a larger number and the more detailed analysis involved a smaller number.
Q And then there is a process, I think, by which you actually then match people for the purpose of doing your comparison; is that right?
A Well, you -- you know, you are going to compare the two populations. I'm not sure what you mean by "further matching."
Q Well, that is my question. Do you match individuals or do you try to match?
A What do you mean? We are doing a cohort. It is a cross-sectional cohort study. We are not doing a case control study or a -- what do they call it? You know, where you have a match control where you take each

individual and trying to match that individual with somebody else.

We are looking at the group as a whole and the groups should be -- the characteristics of the group should be as close as possible. And that is the way we did this analysis.

MR. HOPP: Let's go off the record.

(Brief recess.)

(Defendants' Exhibit 126 was marked for identification by the court reporter.)

BY MR. HOPP:

Q Dr. Dalhgren, deposition Exhibit 126 is a copy of the published versions of your Health Effects studies of the Columbus, Mississippi group; is that right?

A That is correct.

Q And just so we are clear, I think you testified earlier that the Columbus paper is a cross-sectional study; is that right?

A Yes, it is. You know, we went in and examined a bunch of people in point A and then we compared them in a similar situated group of the same point, close in time.

Q Is it is a retrospective mortality study?

A No.

Q And --

A Although there is a lot of retrospective collecting of information about their life-long history of their illness, but it is not a mortality study. None of these people were deceased.

Q Before the break, you went into some detail -- I'm sorry. That is the only copy that I have.

You went into some detail on matches and the idea when you are doing a cross-sectional study, you will match a population against each other as opposed to when you are doing a, say, a retrospective mortality study or a case control study, you are going to want to match individuals; is that right?

A Well, if you are doing a retrospective mortality study, you may not necessarily match individuals. It depends on a study design.

You identify a 29-year-old male who has whatever characteristics you are interested in and then you want to find another 29-year-old male who has all of the same characteristics which -- except the one which would be the exposure and look at health status of the two people.

Q But that is really more an issue of case control studies?

A Case control studies are done that way. It is done with small amounts of people and with rare

diseases. It is a very clinical way to do studies because, as I say, use smaller numbers.

Q Is it your testimony, though, when you are doing a cross-sectional study, it is appropriate to match populations generally as opposed to trying to match individually?

A Yes, that is what we did here.

Q Going back to the Jerome plaintiffs and the Greenville plaintiffs, you used different groups as controls for DNA adducts and for dioxins; right?

A Well, you know, this is not controls. What we are talking about is some convenience sample comparison groups that we used because we had the data, and they were close in terms of some of the variables at interest.

I mean, I have no reason to believe that there is any big age or sex or race or any other factors in PAH adducts; and, you know, we only had this one other group where we had drawn the blood. So I wouldn't call it a control group because we didn't, as I already stated a couple of times, we didn't go out and say here is the people in Grenada, we are going to find a control group for them.

We drew the blood. We had the blood for these others. We ran them together. There were marked

differences between the two groups. That is what we indicated. It is what it is. It is not a formally matched group. It is a convenience sample comparison group.

Q I probably used the wrong words. I know why you gave your answer. It was not what I was looking for in terms of the question I was asking.

Why didn't you use the same people as your convenient comparison group for both DNA adduct and PAHs? Let me explain that a little further.

You testified, I think last time, that you believed that Greenville was an appropriate reference population for Grenada and you were taking blood at Greenville and you used it, and you did the sample runs and then compared them.

Why didn't you go back to Greenville for the purpose of doing DNA adduct?

A That's a good idea. Maybe we should do that. It would be a handy group to look at.

Q But the answer really is that you already had the Jerome data and you were doing that anyway?

A We already sent the samples to Phillips' lab when we drew the Jerome samples and the Homosassa samples. And we were looking for more -- he didn't want to run such a small number because it was a big job to

gear up his lab.
We were waiting to get another PAH exposed group, so we can use it to expand the numbers. So that Dr. Phillips would be willing to do the runs for us. That is why we did it the way we did it.
Q We talked at great length last time about the general decline in dioxin levels in blood in the United States over the last 20 years or so.
Do you remember that discussion?
A Yes.
Q And you indicated at one point that the reason you didn't use sort of general literature values for the purpose of comparing the Grenada population in this case was that the general literature values may be out of date for dioxin?
A Yes, definitely. There is a time factor in our country because there has been efforts to reduce the dioxins in the environment.
Q Have you seen any published literature values which indicates total TEQ in a population of approximately 15? In other words, is there any literature which corresponds with a total TEQ you found in --
A Greenville.
Q -- the Greenville population?

A I haven't looked at the literature with that in mind. So I can't tell you that I have seen it or not seen it. I just don't have an answer to that question.
Q The Schecter paper from 2005 was marked as an exhibit last time, and I think --
A It is at the bottom of the pile here.
Q You are a coauthor on this paper; right?
A Yes.
Q What parts of the Schecter 2005 paper did you actually physically write?
A Oh, I didn't write any of it originally, not to -- Dr. Schecter wrote it. I made a few changes, editorial comments. I don't remember exactly where. I talked to him about it. That was my contribution.
Q Keep that handy because I think we need to refer back to it.
I will show you again what we have marked previously as Exhibit 14 and this is the Axys data.
A I have a copy.
Q Okay. This is the Axys printout for the Greenville group; correct?
A Yes.
Q Now, we talked last time about the notion that for the TCDD value, you have K's represented and you told us what that means.

Is it appropriate, when you have a K value, to assume that the actual number is half the detection limit?
A No, you do that with nondetects.
Q Okay.
A I, frankly, don't know if there is any agreed upon way of handling the K values. I think what I would recommend we do is take their estimated value, realizing that it may be off; but still, if you took half of the value, I think that would be incorrect, as well.
Q And you testified earlier today that a more appropriate shorthand or sort of fudged value for them would be approximately four; is that right?
A Yeah, I mean, that's --
Q And you think it would be appropriate to raise the total TEQ on deposition Exhibit 14 to approximately 20.6? Well, if you add the 4 in?
A Yeah, it would certainly add to it and -- yes, I think that would be appropriate.
Q All right. Now, let's look at Table 5 from your expert report, which we marked last time, which is deposition Exhibit 16; and I have a copy of it right here, so we don't have to fish for it.
Do you just want to look at this?
A Okay.

Q Deposition Exhibit 16 is Table 5 from your expert report in this case. And I want to use both Exhibit 14 and Exhibit 16 for some of these questions coming up.
A Okay.
Q On Table 5, you did not include the data for 2, 3, 7, 8 TCDF; is that correct?
A 1, 2, 3, 7, 8?
Q No. 2, 3, 7, 8 TCDF.
A It's not there, so --
Q And on the Axys table, you have 2, 3, 7, 8 TCDF -- let me find it here.
A Those were all below detection limits.
Q So would it be appropriate to use half of the detection limit as a shorthand?
A Yes.
Q Would that raise the total TEQ?
A No, it would not make any difference. The TEF for that is not that high. I forget what it is. Let me look at the TEF table, but it would make very little difference. I think it is .01 or .1, so -- I wouldn't move it.
Q So half of the limit of detection times the total -- strike that.
Half the limit of detection times the TEF for

2, 3, 7, 8 TCDF would result in a very low number?
A Oh, sure. I mean, half of the detection of it would be .45 for the first one, for example. I mean, it is going to be like .01. It is just not going to make a difference.
Q So it wouldn't raise the total TEQ?
A Not by anything. Let me grab the TEF value. It is .1. The TEF. So .1 times .145 would be .04. It wouldn't make any --
Q I want to return to something that we talked about last time. I am handing you what we previously marked as deposition Exhibit 3. And this is your biomonitoring paper from 2004.
In Table 4, on -- you know what, I got the wrong -- I am barking up the wrong tree, Doctor. I will come back to this issue tomorrow.
We did talk last time about the process that your office went through to translate the values for the mean dioxin levels on Exhibit 14, which is the Axys data to get the values that we see on Table 5 of your report, which is deposition Exhibit 16.
You remember you mentioned there was a statistician in your office who has to apply some sort of statistical program in order to interpret the mean results in order to get your final --

A Yes, she adjusted the values per age.
Q How precisely does that happen? How does it work?
A You use a multiple logistical regression program on the computer and you put in the correction factor of the age and then you calculate the change that that would result in.
Q All right. The total TEQ on deposition Exhibit 16 is 31.43; correct?
A Yes.
Q And that represents the sum of the various TEFs for the dioxins and furans based on a measured concentrate on these 29 individuals; is that right?
A Yes.
Q Now, looking at Table 1 of your 2004 paper, which I just handed you, the biomonitor paper?
A Yes.
Q You represent -- strike that.
You published several different TEF values -- I'm sorry, several different TEQs?
A Okay. What are we talking about?
Q We are looking at Table 4 of the biomonitor paper.
A Okay. Um-hmm.
Q You got TEQs based on PCDD/F. Do you see that?

A Um-hmm.
Q And then there is a TEQ based on PCBs; is that right?
A Yes.
Q And there is a total TEQ?
A Yes.
Q Why, in the biomonitoring paper, do you publish three separate TEQs?
A Well, just for -- someone can do their own calculations. We just make it easier by pointing out the total, the TEQs from PCBs and TEQs from PCDD and Fs.
Q Have you calculated the TEQ based on PBDE/F for the Grenada population?
A That is what is listed here in Table 1, exposed residence, and 29, that is the Grenada people, that is their values. These were not age adjusted.
These were unadjusted values, plus the differences that you see between Table 5 in the report and in Table 1 in this presentation, not plus.
Q When you went to Greenville for the purpose of taking blood samples, did you believe that the chemical plant was a potential source of dioxin?
A I didn't, but Dr. Parant did.
Q Who is Dr. Parant?
A He is a toxicologist who was involved in the

case. And he said, gee, we really should do some dioxins.
And I said, well, I think it's not likely to show anything. Given what they do at that plant, but he wanted to do it. So we did it, as I said this morning and last time, also.
Q He -- you testified, I believe, a couple of times that some PCBs are dioxin-like; is that right?
A Yes, they have a TEF. We talked about that.
Q We talked about it generally, but what specifically does that mean? What does it mean to say -- what is a PCB?
A It is a benzopyrene with chlorine attached, similar to a dioxin and furans. They are all cousins, if you will.
Q But PCB are polychlorinated biphenyls?
A Yes.
Q They are, in fact, different chemicals from dioxins; right?
A Yeah, they are a different class. The difference is a dioxin has two oxygens between the two benzene rings and a furan has one, whereas this doesn't have any oxygen between the benzene rings.
Q So when you say a PCB is a dioxin-like, do you mean it is dioxin-like in its chemical structure or do

you mean dioxin-like in its effect?
A Dioxin-like in its effect. That is where you give its toxicity equivalent factor and it is what they will do to get that is to do an in vitro test as to how much the particular congener PCB stimulates the AH receptor.
Q Okay. So they do an in vitro test, find out how much of an enzyme is generated between the PCB congener is combined with the -- binds with the AH receptor?
A Not the enzyme. When the PCB binds to the receptor, how much of a reaction that can be measured with enzyme amounts or other ways of assessing the activity of the stimulation of the receptor but, yes, in general, that is how it is done.
Q Are all PCBs dioxin-like?
A No. Only the so-called coplanars.
Q And which are those?
A The ones that have a certain steric structure and there -- those are the ones that tend to be the flattest and that is because of the steric inhibition of where the chlorine atoms are attached. There are several places on the benzene ring where there can be chlorine attached, that is what distinguishes the different PCBs.

And there is a relatively small number that are coplanars and stimulate the age receptor. And there are a few that are not totally coplanar, but they still stimulate the receptor, although weakly.
Q There are 209 congeners of PCB; correct?
A Yes.
Q How many congeners of dioxin have been identified?
A It is about a hundred -- I think 112. I forget the number, something like that.
Q Of the 209 dioxin congeners -- strike that.
Of the 209 PCB congeners that have been identified, how many have been identified dioxin-like?
A Let me identify the table that, that I was looking at a minute ago. I didn't put in this table -- I don't remember from memory. It is probably six or seven that have TEF that has been calculated for them.
Q So a handful?
A A handful.
Q Of the 209?
A That's right.
Q Now, you have included in your report references to literature on PCBs; is that right?
A Yes.
Q Is it your opinion that the plaintiffs in this

case were exposed to PCBs?
A Not from Koppers, probably. Not unless there was some exposure that we have not identified yet.
No, I did not include the literature on that for that reason.
Q Why didn't you include it?
A Because there is an overlap. The toxicity of PCBs overlaps with the toxicity of the dioxin and the furans.
In other words, the entire class for polychlorinated biphenyls, two benzene rings, there is a lot of similarity of the diseases they cause, like chloracne immune system impairment, neurological changes, cancer.
So it is just, in my opinion, one would not be surprised to see a lot of the literature that is -- a very large literature there to enlarge our understanding of the toxicity of this class of compounds.
That is why, for example, in dioxin meetings, every year there are numerous papers on PCB. It is not a PCB conference, but PCBs are dioxin-like. So their toxicity is relevant when we are talking about this class of compounds.
Q I am having a little trouble understanding you.
There is only a handful of PCBs that have TEF;

correct?
A Yes. That's right.
Q And is it those PCBs which -- and those are the PCBs that are dioxin-like?
A Yes.
Q So when you say there is an overlap in the literature, is there not overlap in the entirety of PCB literature with dioxin, of dioxin or is it an overlap of dioxin-like congeners of PCBs?
A Almost all of the studies of the PCBs have focused upon the toxicity of the dioxin-like PCBs. That is what they are looking for.
Now, there is also now some recent, in the last few years, research where the non-dioxin-like PCBs may have some additional toxicities that are not shared, at least not shared in a powerful way with the other dioxins.
But, generally speaking, you know, 98 percent of what we are talking about with PCBs is their dioxin-like toxicity; therefore, the toxicity of a PCB exposed person is similar to the toxicity that you see from all of the other dioxins. So it is because of the end points in the PCB population that we bring them in for the discussion.
Q Now, we are going to look at some of these

studies most likely tomorrow. But isn't it true that there is not really only one or two PCB congeners that have been identified as posing a risk for breast cancer?
A I would say that they -- that that is the leading thing that the -- that some of PCBs are more estrogenic in their effect and have more an ability to stimulate the age -- not only the age receptor, but the estrogenic receptor. And so I would agree with you that there are certain PCBs that have that effect.
Q PCBs are identified -- strike that.
PCB congeners are identified by number; right, PCB 1 --
A 1 to 209.
Q As opposed to dioxin congeners, which are identified by a chemical structure; right?
A Yes, and that is because there is -- the PCBs, it depends on the relationship of the chlorines can change the chemical nature of the molecule, whereas with the dioxins, you don't have that many of the -- all you have to worry about is how many chlorines are on the two benzene rings. You don't have to worry about what orientation the chlorine has relative to the other chlorines.
Q Don't some of the PCB papers say that it is really only PCB -- I think it is 153 which is identified

as being a risk factor of breast cancer?
A You know, I have not looked at that literature. I don't recall off hand.
Q That's okay. We will get it tomorrow.
In any event, none of the plaintiffs that you are aware of in this case was specifically exposed to the dioxin congener or the dioxin congeners, which you have identified of increasing the risk of breast cancer; right?
A Yes. I am not asserting that the PCBs were the cause of her breast cancer per se. I mean, what I am saying is that there is dioxins in PAHs and benzene and probably some of the other chemicals that you have identified are the cause of her breast cancer.
Q Well, we will come back to the literature tomorrow. I want to jump briefly to the Hubbard procedure or you called it the detoxification procedure?
A Yes.
Q And this is the -- again, for context, the procedure that is being applied to the New York City firefighters; correct?
A Correct.
Q And I believe you told me that the technique had been used to enhance the elimination of similar compounds in the body; right?

A Yes.
Q And part of the procedure involved extensive sweating, sitting in the sauna; right?
A Yes.
Q Does the sweating itself; that is, does the sweat help to express out the dioxin?
A Yes. Some of the lipid soluble chemicals, such as dioxin and PCBs and that class, which are highly lipid soluble, are secreted under the skin and excreted from the body in that way.
Q Have you made any effort to -- well, strike that.
What is the chemical composition of sweat?
A Well, I mean, sweat is basically -- depends on what -- if you are talking about what is in all sweat, it is basically some water and some minerals.
Q Water and solvents?
A Water and solvents is the main constituent.
Q And dioxins and furans are not soluble in water; right?
A That's correct.
Q And given that dioxins and furans are not soluble in water, can you explain to me on the basis that they would be excreted from the body would be a sweat?

A What that is is the skin oils. What you do is increase the amount of the skin oil that is produced and we measure PCBs -- for example, I think I told you last time, in the skin oil of patients undergoing this detoxification procedure.
Q All right. Did you measure dioxins in skin oil both before and after they started the procedure?
A No, just during the procedure.
Q And is there a reference value that you were able to use to identify whether they had more than the normal amount of --
A Well, all we noticed was the amount of PCB per gram of fat was high in the skin oil.
Q In the skin oils?
A It was higher than it was even in the patients' blood fat at the time.
Q And you have published that data; that is, the data on skin oil measurements for dioxin?
A It was published by Cedrick Trusk (phonetic) in the paper that I told you about.
Q That was the paper from the '70's or '80's; right?
A Yes.
Q Cedrick brought some woman from --
A -- Yugoslavia.

Q Here to the U.S. for treatment?
A Right, for treatment.
Q But for the firemen, you published a paper on the firemen?
A We did. We did blood dioxins and PCBs and PBDEs, as well, both before and after.
Q But have you reported the skin oil measurements?
A No, we did not do the skin oils of the firefighters.
Q I'm sorry. I misunderstood.
A We did blood values.
Q I'm sorry. I think I misunderstood one of your prior answers.
You were talking about the -- your basis for assuming that dioxin can be excreted out of someone's body in sweat. And in your answer, you mentioned skin oils. I thought you said that you tested the firemen's skin oil?
A No, we just did their blood.
Q I misunderstood. You did not check the firemen at all for skin oil?
A We did not do any skin oil testing on anything.
Q For the basis that dioxin can be expressed in skin oils is the Cedrick paper?

A Yes, all we can really say is that the values came down significantly in the blood in both this lady in Yugoslavia and in these firemen that we have tested so far; but I -- based on my experience with the Yugoslavian lady, I think the skin oil is one of the routes of secretion; but there are others, as well.
Q And other one is feces?
A Feces is probably the main way, but without a full study, you know, where we spend money doing before and after in all of those different organ systems, plus even doing some measurements during the process, as well as at the end, you know, it is -- I wish I had the resources to do that.
Q One of the things you could have done and had resources to do is stool samples both before, during, and after?
A Absolutely.
Q Blood samples both before, during, and after?
A That's right. And sweat samples.
Q Skin oils?
A There are -- there are some metabolites of PCBs and dioxin where they get attached to certain molecules, like glucuronide and hydroxyl groups, which make them water soluble and come out in the urine; and that may be a root of exposure that we speed up with our process.

What we can say is that we are doing, you know, we are making some progress, but the details about how the body is doing it, we are not able to say.
Q You just know that the blood levels appear to be decreasing?
A The blood level drops and the patient experiences a significant change in how they feel. We have not published this yet, but we have big changes in some of the neurologic test we do and some of the blood tests change. So we know that we are doing something physiologically.
Q Is there any way to determine whether the changes in the way the patients feel is due at all to the placebo effect?
A Well, there may be a placebo effect. It is pretty hard to do a sham treatment with this type of thing, but I mentioned we have objective tests reaction time, balance, pegs filling a hole.
Q Neuron testing?
A Neuron testing battery that we have been using. We have been using it on the firemen before and after.
We have been trying to get the data to publish it, but somehow have not been able to get the data pulled together. We -- in the ones that we looked at, there is marked improvement of the objective test.

Q The detoxification treatment involves saunas and massages and a special diet; right?
A Well, it is niacin, high doses of vitamin B3 to mobilize fat, exercise; cardio exercise; sauna; vegetable oil; cold pressed oils. Massage is not part of it.
Q Okay.
A Although we have sometimes done that, but it isn't rigor.
Q But if I was suggested to a regimen that involved saunas and exercise and diet, I probably would feel better than I do.
Do you think that is what you are picking up, that these people are being subjected to these -- this treatment and the objective case is that they are feeling better in the regimen that is being administered as opposed to the lower dioxin levels?
A I think it is more than placebo effect. There is more a physiological change that occurs in the body.
Q You tested the firemen's blood after --
A After the detox.
Q Not before?
A We did it before and after.
Q Is the pretesting concentration reported in deposition Exhibit 6?

A No. This is the first value. So these were the prevalues, I believe.
Q Okay.
A We published them in the final paper that we actually presented in Berlin. This was just the original abstract that we submitted. We didn't have the postvalues at the time we put that together.
Q So the postvalues have since been published --
A They are available in the website. And if you would get the organohalogen -- what is it called? Organohalogen --
Q Compounds.
A -- compounds. The post-meeting publication does include the postvalues. Didn't I give it to you during the last deposition?
Q No.
A Because we wrote up the posttreatment values.
Q What you gave me at the last deposition was the 2000 Schecter paper and the recent --
A We have a paper that we -- the paper we presented, actually, included the pre and postvalues.
Q If possible, tomorrow, if you can bring that with you, I would appreciate it. I would like to look at that one.
A All right. I can actually put it on -- I think

I got it here. I can put it on the CD. Let me see if I got it here or not.
Q Can I ask you questions while you are looking or would you focus on what you are looking for?
A Let's see. I have a copy of the paper here. Let me open it up and see if it is the right one.
Yeah, here is the pre and postvalues. Yeah.
Q Actually, if you can -- I don't know if I can print it out. If you can bring a printout with you tomorrow, I would appreciate it.
A I will bring a printout tomorrow.
Q You also administered a drug or medication to these firemen; is that right?
A Niacin. It is a vitamin. High dose vitamin. We use pharmacological doses, more than you need, for vitamin purposes; but niacin has been used for years to lower cholesterol.
That is why it is generally available in large doses. It does not require a prescription. You can buy it on your own, over the counter.
Q Sure. I thought I saw a -- something else mentioned. Okay. So the only thing they were administered was the niacin?
A And the cold pressed oils.
Q For the purpose of your dioxin analysis, you

used ERGO Laboratories in Germany; correct?
A Yes.
Q Is there a laboratory in the United States that does similar work; that is, evaluates blood samples for dioxin levels?
A Oh, I think there is probably a lab or two that does it. I'm -- I am aware of Research Triangle Labs, and I used them once.
And then there is some other lab that I saw recently and somebody used for PCBs. I think it was in another little lawsuit. I don't remember the name of that lab.
So there is a couple of labs that, I think, that holds them out to do dioxins in the United States.
Q Why do you use ERGO Labs?
A Well, I relate to the experience that I had with the Research Triangle Labs, I sent them some blood samples of patients that had been exposed, I thought, to dioxin-like materials and they came back all nondetect.
Gee, everybody has got some in their blood. Why didn't they find it? I sent the same -- very same patients to ERGO and got all positive reports. I lost faith in the lab. And I said I am not going to use them again.
Q Is there a consensus standard -- is there a

consensus standard of performing tests of human blood for dioxin?
A When you say "consensus standard," if there is internal --
Q Is there a consensus standard in performing the test for dioxin?
A Not being a laboratorian who does those analysis on a regular basis, I would say I am not certain. I believe there is generally a protocol that is followed, but there may be some variations.
The important thing is whether or not the laboratory participates in an external check sample program where they get unknowns and analyze them and send them back to see whether they got them properly scored or not.
And both Axys and ERGO engage in those types of external check sample programs.
Now, I don't know about other labs, as I say. Midwest Research Lab is set up to do dioxins and PCBs and then quit after a while because they couldn't get the technique done properly. I can tell you that it is incredibly difficult to do dioxins, furans, and PCBs, specifically the coplanars that are present in small concentrations, relatively speaking.
We are talking about parts per trillion

analysis, parts of quadrillion analysis. And so in order to do the laboratory work properly, you have to have incredibly stringent laboratory controls.

Without going into detail, it costs hundreds of thousands of dollars, if not millions of dollars, to set up a lab, and then it costs an enormous amount to run it.

That is why each test is so expensive and darn few labs in the world can do it properly. Dr. Papke, at ERGO, does samples all over the world, not just Europe or the United States. He does it from Asia, South America.

If somebody wants to do a PCB or a dioxin, they have to look long and hard to find a lab that they can trust. And that is why Papke is so busy with the work because he has earned the trust of scientists all over the world.

Q Now, has the U.S. EPA endorsed any particular test method for evaluating dioxin congeners in human blood?

A You mean is there an EPA or recommended method or approved method?

Q Right.

A I believe there is, but don't know what it is.

Q Do you know if the World Health Organization or

NATO have different approved test methods?

A Different than EPA?

Q Right.

A I don't know. EPA doesn't do the test themselves. CDC has a laboratory in Atlanta where they do dioxin tests; but they wouldn't do them for private citizens.

Q Dr. Papke, on his report, which is deposition Exhibit 17, identifies various accreditations he has received. You see?

A Yes.

Q Do you know if any of those accreditations actually cover the test method for identifying dioxin congeners in human blood?

A I am not familiar with the accreditations that are listed here. I am just simply familiar with his publications and his results and the consistency of his results.

Q I know we may have covered this, but cigarette smoke contains dioxins?

A No. Dioxin has PAH in it. Cigarettes, if they contain dioxin, I have not read about it.

Q When -- if a person burns trash, would you expect it to give rise of a higher level of dioxin in their blood?

A It depends on how much they burn and how much smoke they breathe. Not many people burn garbage in their house. I suppose there are still people that burn garbage.

Q Does that create dioxins, the smoke?

A It would. Burning trash creates dioxins. That is one of the reasons why incinerators for both garbage, trash, and hazardous wastes are controversial because you do not want to create dioxins, which you could, if you don't have a high enough temperature and you are burning.

Q All right. Explain to me the concept of TEFs.

A Well, I -- as discussed earlier, you take the chemical in question and you test it to see how potent it is in terms of stimulating the AH receptor.

Q TEF is a toxicity equivalency factor?

A Factor, and that is used to determine the toxicity equivalent quotient or quantity.

Q The TEF is really a number that is in relationship to the toxicity 2, 3, 7, 8 TCDD?

A TCDD is considered the standard around all of which the -- all the others are measured.

Q TEF for 2, 3, 7, 8 TCDD is one?

A Yes.

Q And there is one other dioxin congener that has

that -- also has a TEF of one?

A I think you are right. There is one more that has one. I don't have it in my mind here, but it depends.

You know, there are different TEFs that have been published, quite different authors, so the ones I used, I think are the EPAs and they are the most conservative.

Q Okay. The World Health Organization actually does have another set of TEFs?

A They have a slightly different -- there is a lot of overlap, but there is some slight differences, yes.

Q Does NATO have another set of TEFs?

A I don't recall. The 1, 2, 3, 7, 8 pentaCDF has a -- using this particular TEF of .5, which is the next most potent one in this particular set of toxicity equivalents.

Q Other than the one other congener of dioxin that has a TEF of one, all of the other 200-some dioxin congeners have a TEF of less than one; correct?

A Yes.

Q In which you get the -- TEQ, I know we talked about this subject, but just so we cover it, what do you identify the TEQ for a given sample -- blood sample if

you were to take the TEFs?
A Multiply them by the concentration that you found.
Q Multiply them by the concentration and then just add all of resulting numbers; correct?
A Yes.
Q Your TEF -- strike that.
If when you do your evaluations, you leave congeners off your table, you are going to get a TEF that is -- I'm sorry, a TEQ that is lower by the sums that you are missing; right?
A Yes.
Q Do you agree with the following statement: There is a wide discrepancy between the draft dioxin risk characterizations of the U.S. Environmental Protection Agency and those of respected public health agencies, such as the U.S. Agency for Toxic Substances and Disease Registry, the joint United Nations Food and Agriculture Organization/World Health Organization Expert Committee on Food Additives and the European Commission Scientific Committee on Food?
A Wide discrepancies.
Q Yes, a wide discrepancy on their draft dioxin risk characterizations?
A As I said earlier, there is slight

differences -- I noticed those tables that I read to you are of the WHO TEQ?
Q So your report does use WHO?
A I thought it was EPA, but if it is WHO, there are differences, but I don't think this is wide discrepancies. I think it would be an overstatement to call them that way.
Q Do you agree with the statement that U.S. EPA used very conservative assumptions in policy positions to arrive at a dioxin risk characterization that is 100 to 1,000 times more conservative than those of the other three agencies that I just mentioned?
A 100 to 1,000 times more conservative in terms of them recommending a lower exposure?
Q Right.
A I don't have an opinion about that.
Q Do you agree with the statement that toxic equivalency factors were develop to facilitate risk assessment and regulatory control of dioxins and dioxin-like compounds, but their usefulness is severely limited?
A No, I wouldn't agree with that. I think they are very, very useful in terms of necessitating the toxicity of this complex mixture and that arguing about toxicity -- I think what these toxicity equivalents

usually do is underestimate the risk, not overestimate it. And that is just based on my own experience.
I do believe that they were derived for regulatory purposes so that they had a number that they can enforce.
But in terms of it being 1,000 times or even 100 times out of sync with reality, I don't think there is a shade of evidence to support that. In fact, quite the opposite.
I think, given the fact that people are exposed not only to dioxins, but to all of the other chemicals that have parallel effects, for example, estrogenic effects, stimulation of the age receptor, that we need to start looking at the synergistic effects.
And it may well be that the current levels of dioxins are exerting a tremendous adverse effect on the population. And then by no means can one extrapolate from some animal model where you look at one chemical at a time and say, oh, this chemical is safe because I was able to feed it to this rat; and nothing happened to this rat; or its offspring; or its brain chemistry; and, therefore, that is a safe level. That's not the real world that humans are in.
They are exposed with this chemical along with 50 other chemicals that have actually similar adverse

effects; particularly in the brain or the immune system.
So I think that, you know, right now, I don't think they are being proactive enough in looking at mixtures and their health effects.
In fact, there was a paper given in the dioxin 2004 meetings by the former head of one of the branches of Health Canada where he made this exact point where we are chasing one chemical at a time and failing to assess the very important interactions of all of these chemicals.
In fact, I brought along a paper this morning that I found that addressed this exact issue. They looked at some mixtures of PAHs and they showed that when you add dioxins to that mixture, you get a synergistic effect in terms of the toxicity end point that they were measuring, which is the K lack system. That is the way they are assessing the dioxin-like behavior.
And when you have PAHs and low level of dioxin together, you get not just an additive one and one, you get a synergistic effect. An effect of three or four, five times of what they would expect.
They made it a point expressing the toxicity using TEFs would not have predicted the effect that they saw in this mixture. So I think arguing about the

safety is too great with the level of exposure that we are talking about.

By the way, EPA risk assessment suggested that we need to lower dioxin levels even further was based on research which shows that the effects were occurring in animal test systems with the single chemical at lower levels than we had previously thought; and that is why they made this recommendation about continuing to lower environmental exposure even further than they already are.

Q Well, to have a synergistic effect between two chemicals or for two chemicals to have a synergistic effect, each of those chemicals have to cause the effect by itself; right?

A Not necessarily. I mean, that is usually the case, but there are situations where either one at the dose level that we talk about have any effect, but the two together do have an effect when they are present together.

Q In order to have a synergistic effect, each chemical has to produce the end point in question at some dose level; is that right?

A No, not necessarily because the mechanism by which you get this synergy can be due to a simulation of an enzyme that metabolizes the other chemical to the

toxicity intermediate and then it has its effect.

And it is not because chemical one, which stimulates the enzyme system, would go on and have that other effect; but it would have the net effect of having a synergistic effect. Asbestos and cigarettes are an example.

Q Is there a difference between promotion and synergy?

A Yes. Promotion has nothing to do with synergy.

Q All right. Let's talk about synergy.

If, for example, you have a substance which is not known to cause breast cancer in rats at any dose level, are you saying that it is possible that that substance can have a synergistic effect with some other substance where it --

A Yes, it can stimulate the enzymes that can produce the toxicity -- toxic intermediary. It will not cause breast cancer, but it will cause another chemical to have a greater propensity to cause breast cancer by stimulating the metabolism in that tissue. The enzymes that metabolizes PAHs to the toxicity intermediaries are stimulated by dioxins present.

Q Now, you have given us today, this morning, I think we talked about this off the record, but you have given us a set of lists of different articles; correct?

A Yes.

Q They are broken down by subject area; is that right?

A Yes.

Q And some of these articles are articles which are cited in your report; is that right?

A Yes.

Q And some of the articles contain on -- listed you gave today are not cited in your report?

A Yes, there are some new ones. Particularly on breast cancer, there are quite a few new ones and adducts, quite a few new ones.

Q Last week during Dr. Sawyer's deposition -- a week and a half ago during Dr. Sawyer's deposition, did you talk to Dr. Sawyer while he was --

A No, I didn't. One of my staff members, I think, spoke about sending us some papers about breast cancer and PAHs and dioxins.

Q Did you or your staff send to Mr. Prudhomme, during Dr. Sawyer's deposition, a list of articles on various topics related to this case?

A Yes.

Q And does that list contain all of the new papers we see represented in the stack of lists that you gave me today?

A Well, I think I added a few papers since that week and a half ago.

Q Even since that week and a half ago, there are new papers in this biography?

A Yes, I gave them to you in one of the CDPs presented this morning. So you don't have to go to the library and find them all.

Q I appreciate that. You do have, I think, a table entitled Mixture; is that right?

A Yes.

Q Are those your references on this issue of synergy?

A Well, it certainly addresses -- yes. I don't think this most recent paper that I just mentioned to you is on here yet; but this talked about the fact that when you are looking at mixtures, you see different effects than you might have expected looking at a chemical by itself.

But, yes, this is mainly -- the interaction business, the business of mixtures, that is slightly different that synergistic. Although there are several -- couple of papers that have to do with synergy. Most of them have to do with just the issue of mixtures.

Q All right. So do we have a separate

bibliography of your separate list of articles that relate to synergy?
A Well, like I said, I brought one paper this morning, which is an additional paper that I --
Q Can you give me a citation for that one? So we can get it on the record.
A Sure. Let's find it.
Q This one is entitled Evaluation of Mixture Effect in Crude Extraction of Compost Use CLAUS Bioassay and HPLC Fractionation?
A Um-hmm.
Q Yes?
A Yes.
Q And the lead author is Suzuki; correct?
A Yes.
Q Publication is Environment International 2004; right?
A Yes.
Q Can I have this copy?
A Yes.
Q Let's look at that and we will talk about that tomorrow. You have cited in your report interaction profiles that have been published by EPA?
A Yes. They address the problem -- it is not an EPA --

Q I'm sorry.
A They are devoid of very much data. They basically, all of them, just talk about the problem and the need for studies and recommendations for studies and so on; but they are really very deficient in terms of information; but they highlighted the problem that we are talking about.
Q But other than in the Suzuki paper or the papers identified on the lists that you gave us, are you aware of any other papers that specifically address the synergistic effects of PAHs and dioxins in causing human breast cancer?
A No. Well, yes, there is a paper which, I think, this is on that list, that talks about if you expose a rat to dioxin during pregnancy, then that rat's offspring is most likely to get breast cancer on exposure to PAHs. You get a higher rate.
There is some kind of change that is induced in the fetus that alters their suseptibility of breast cancer.
Q That is in rats, then?
A Yes, it's a rat study.
Q Are there any studies that you are aware of that demonstrates a synergistic effect between PAHs and dioxin for the induction of human breast cancer?

A I don't know how you do the study. Yeah, I am not aware of one.
Q 2, 3, 7, 8-TCDD is a known human carcinogen; correct?
A Yes.
Q Who considers 2, 3, 7, 8-TCDD to be a human carcinogen?
A The National Toxicology Program has rated it as a definite human carcinogen. I'm not sure about some of the other agencies.
Q IARC, International Agency for Research on cancer?
A Yes.
Q Headquartered in Lyon, France?
A Yes.
Q And they publish monographs on an occasional basis to discuss substances that are known human carcinogens or suspects them?
A Yes, they classify them.
Q Has IARC classified 2, 3, 7, 8-TCDD as a noncarcinogen?
A I don't recall.
Q Is 1, 2, 3, 7, 8-PeCDD a known human carcinogen?
A I don't think so, no.

Q Is 1, 2, 3, 4, 7, 8-HxCDD a known human carcinogen?
A No, I think all of the dioxins are, as a group, basically felt to have the potency of the TCDD per the TEF and that potency is its ability to not only cause cancer, but other health effects; but it is my understanding that that is what we are talking about is cancer-causing capacity.
Q But the fact is that none of the other congeners of dioxin; that is, none of the congeners other than 2, 3, 7, 8-TCDD has ever been identified by IARC or the NTP or anyone else as a known human carcinogen; correct?
A I don't think anybody has done any studies to specifically look at that question. So as far as I know, there is no data on that question.
Q So despite -- strike that.
Is 1, 2, 3, 6, 7, 8-HxCDD a known human carcinogen?
A Like I say, I don't think any data has been generated on that specific chemical per se, except indirectly, as I discussed earlier, in terms of assessing its TCDD-like qualities.
Q Has any national or international body ever classified 1, 2, 3, 6, 7, 8-HxCDD as a human carcinogen?

A I think I answered that question already.
Q And the answer is no?
A The answer is no one has ever actually done the study because it is not necessary. I mean, we -- there is no point in going -- making a bunch of that chemical and feeding it to the animals.
We know that it is going to have that effect based on the TCD-like behavior. So, I mean, I suppose someone can do it, but no one has. Just probably because it is not an interesting question.
Q So the classification does not exist; correct?
A I think I already answered the question.
Q Is 1, 2, 3, 7, 8, 9-HxCDD a known human carcinogen?
A It is the same answer. It has not been studied, as far as I know, as its individual chemical by itself.
Q Is 1, 2, 3, 4, 6, 7, 8-HpCDD a known human carcinogen?
A Same answer.
Q Is OCDD a known human carcinogen?
A Same answer.
Q Is 1, 2, 3, 7, 8-PeCDF a known human carcinogen?
A Same answer.

Q Is 1, 2, 3, 4, 7, 8-PeCDF a known human carcinogen?
A I think I can save you some time. The rest of that list is the same answer for all of them.
Q Same answer for every other dioxin congener?
A As far as I know, they have never been looked at by themselves. No one has ever synthesized them or used them alone to try to poison some animals and see what happened to them.
Q But those studies could be done; correct?
A A lot of studies could be done. With limited resources, you don't do every single study in the world. We have known that if they have TCD.
Q What is an AH receptor?
A Arylhydrocarbon receptor, it is a receptor that is present in every cell in the body. It controls -- when this receptor is stimulated, it controls the growth of the cell and the turnover of the cell. So it is an important receptor.
Q How does the concept of an AH receptor play into the study of dioxin?
A Well, it is felt that most of the adverse effects of the dioxin and dioxin-like materials is mediated through that receptor.
Q How is that mediated?

A When that receptor is stimulated, it has the adverse effects that we talked about. In fact, it is sometimes referred to as the dioxin receptor because it is very specific for this class of compound.
Q When the age receptor is actually a physical structure on a cell?
A All receptors are actual physical structures on a cell.
Q And the dioxin molecule binds to the age receptor; is that right?
A Yes. And, thus, stimulating it to do what it does and exactly what it does is fairly complicated, goes into the -- causes the release of another compound that then travels to the -- where is it, to one of the other structures in the cell and has its effect there.
Q It has the effect down the line of inducing enzymes; correct?
A Inducing enzymes and also inducing regulatory features of the cell, which is why it is so dangerous because it affects that cells signaling of that growth and regulation apoptosis and other critical stages of cellular function.
Q On what cell structure would we find the age receptor not on the cell memory? How many naturally occurring substances and other synthetic chemicals are

capable of binding with and activating and deactivating an AH receptor?
A The PAHs stimulates the age receptors, they are weak compared to the dioxins, but they do have that capacity. The paper I just gave you talks about that to some degree because the K lack system is one of the ways you can see how stimulated the age receptor is.
Q PAHs and what else?
A Oh, there are a few other things that have been identified.
Q Can you name them as you sit here?
A I am trying to remember. I can't remember right this minute.
Q If an AH receptor antagonist is bound to the AH receptor, does that mean that dioxin cannot then bind to that receptor?
A Yeah. If you had antagonistics to block the receptor that -- so that it couldn't be stimulated, then you would block the effect of the dioxin.
Q Does dioxin need to bind to an AH receptor in order to have a toxic effect?
A Well, that is generally what people believe. Although there are a couple of other mechanisms that have been talked about that they don't seem to be as nearly as important, but other effects that it can have.

Q Do you know what those other mechanisms are?
A I don't recall right off hand.
Q Dioxin is not, by itself, chemotoxic; correct?
A Yes.
Q Is it something that -- PAHs are, by themselves, chemotoxicity?
A Yes, they are very much so. That is their main thing about the PAHs is their ability to bind to DNA and, therefore, cause destruction of signaling and reproduction, faithful reproduction of the DNA.
Q Has the mechanisms for action of -- action for dioxin been studied in mice?
A Yes.
Q Have they been studied in other species?
A Yes.
Q Which other species?
A Guinea pigs, rats, dogs, monkeys. Probably dozens of other studies, I'm sure, have been done in all kinds of species.
Q Have they been studied in humans?
A Yes.
Q Have there been any papers published that TCDD gives rise to enzyme induction in humans?
A Yes.
Q Can you cite one?

A Not off the top of my head. I have to go and do some research that stimulates a variety of enzyme systems of the body in humans.
Q What else binds the AH receptors?
A You asked me that already.
Q You're right. Strike that question.
What is the evolutionary function of the AH receptor?
A I don't remember. I think there was one, but I am not sure what it was.
Q Do you know if anybody really knows the answer to that question?
A I don't know.
Q What are the naturally occurring ligands -- that is L-I-G-A-N-D-S -- for the AH receptor, these are naturally occurring substances?
A I don't recall. I don't remember.
Q Do you know what a knockout mouse is?
A A knockout mouse is a mouse with certain genetic factors that cause them -- for example, you can have knockout mouses with different things knocked out; but one of them that you are probably referring to is a knockout mouse that does not have an AH receptor.
Q You can breed a mouse that does not have an AH?
A And they are not subject to the anti-toxicity.

Q How do those mice do generally?
A I don't know.
Q Do you know how healthy they are?
A No, I don't.
Q Has enzyme induction been studied in the Seveso, S-E-V-E-S-O, cohort?
A Enzyme induction, looking at liver enzyme activity, for example?
Q Yes.
A I do believe that was done. It wasn't done on them. It has been done on others.
Q Has it been studied -- has enzyme induction been studied in NIOSH worker cohorts?
A I think that there was something -- I think that was done on that group, but I am not certain. I would have to check the paper.
Q Are there molecular differences between human and mouse AH receptors?
A I don't know.
Q Do different strains of mice react differently to identical doses of TCDD?
A Different strains react differently, yes.
Q Is it true, compared on -- compared to the data, is it true that compared to the data on mouse AH receptors, that human AH receptors appears, under free

cell conditions, at least to have a several fold lower affinity for TCDD?
A The mouse receptor?
Q Compared to the mouse, the human has several folds lower affinity for TCDD?
A I don't know below that.
Q And is it true that some data suggest that the human AH receptors may be many times less sensitive than the mouse AH receptor in eliciting a response?
A Many times?
Q Many times.
A I don't know the answer to that. I know there are differences, but I don't know the quantitative differences.
Q Are many AH receptor modulated against regulated in a species cell and developmental stage -- strike that.
Are many AH receptors modulated against regulated in a species cell and developmentally stage-specific manner?
A I think you got a compound question there.
Q I do?
A There are lots of issues. Probably the answer is yes, but I don't know what it means exactly. Why don't you break it into pieces.

Q Why don't I suggest, does the answer to that question discuss that a molecular and cellular pathways leading to a particular toxic event are extremely complex?
A That, I think, is a fair statement. The complexity of the toxicity of these compounds is great.
Q All right.
A And like I mentioned to you earlier, that the exposure in uterine altered the susceptibility of the offspring in life to develop breast cancer.
I mean, that doesn't happen if you expose the animal to TCDD when the animal is mature. So there are differences in timing, just as an example, as we passed by the question complexity, it is very complex.
Q Is it true that biochemical and biological outcomes of TCDD exposure can be modulated by numerous other proteins with which the AH receptor reacts?
A Yes, there are a lot of interactions that go on.
Q Is it true that even for TCDD, many unanswered questions still exist regarding how it causes cancer in mice?
A Yes, it is still not certain -- the mechanisms are debated as to how a nongenotoxic chemical can cause cancer. But as I said earlier, I think most thinking is

that it has to do with this regulation of growth, and apoptosis. There is two functions that are shown to be interrupted and affected by dioxin.
And just to amplify on the second point, apoptosis is the normal function of the cell. When there is an abnormality of any kind, the cell says, okay, we have an abnormal DNA, abnormal functioning part of the cell; we are going to kill this cell and in an orderly fashion.
And so that, basically, it turns on a mechanism for cell death. And it is an orderly process. And all of the components of the cell are then reused by the body, you know, as opposed to necrosis, which is an adverse cell death caused by, for example, when you have a heart attack, the cells die.
It is not a -- apoptosis, it is not an orderly progression of death and it has adverse consequences as a result of that. You know, the cell doesn't renew itself. You don't get a new cell to replace it necessarily.
So apoptosis is an important mechanism to get rid of abnormal cells, but maintain the optimal function of the organism. When apoptosis is interfered with dioxin, allowing cells that should die or be killed off in an orderly fashion by the body, thus, you got an

abnormal cell that is allowed to live and keep going and produce.
That may be one of the mechanisms, but there is no agreement as to what the mechanism is where at the mutagenic agents like PAHs. Everyone thinks they understand that the mechanism is fairly straightforward.
You damage the DNA in sufficient amounts and quantities and places. Cancer is one of the consequences that does occur from that genetic damage.
Q The point that you are trying to make is there is still a lot of unanswered questions about how TCDD caused cancer in mice?
A There is still unanswered questions about everything in medicine, but TCD dioxins have a lot of questions.
Q There is lot of unanswered questions about how the mechanism works whereby TCD causes cancer in mice?
A Yes. And, you know, how many billions of dollars have been spent trying to figure out cancer in any setting anywhere, anymore, anyhow. We know preciously little in spite of the billions we've spent on research. It is a complex business.
Q Is it true that the --
MR. PRUDHOMME: Is this a good --
MR. HOPP: I have a couple more questions.

Q Is it true that the mechanism by which TCD may cause cancer in humans is even less well-understood than what we understand about mice?
A About mice?
Q We just established that the mechanism by which TCD causes cancer in mice is not well-understood?
A Yes.
Q Is the mechanism by which TCDD may cause cancer in humans even less well-understood?
A We usually learn about mechanisms by doing animal studies. If we learn to -- learn from -- animal studies allows us to intelligibly design treatment and preventions in humans.
And insofar as we don't have a mechanism, it is very difficult for us to develop treatments. I am not -- you know, I didn't prepare myself for the issue of the mechanisms by which dioxin causes cancer; except in my normal, natural reading, I picked up a few pointers about it; but I don't think we know the mechanisms of most cancer-causing agents and most cancers are of unknown cause.
So there is a lot we have to learn. That does not mean we cannot identify certain high risk situations of more cancer is occurring as a result of certain exposures. We can certainly do that.

There seems to be very little controversy of cigarette smoke associated with cancer. We don't necessarily know all of the details.

Why cigarette smoke causes cancer in one person and not the next. They both smoke the same amount, live in the same town, same genetic background in terms of genetic background. One gets it; one does not. What is going on?

Q In humans, we have the basis of observational studies to try to isolate exposures and try to see the increase incident?

A Yes.

Q Do you accept the notion that the dose response curve of toxic effect of dioxin exposure in humans is not linear?

A I don't know of any data on the question of a nonlinear dose response for cancer induction. In other words, as I understand it, the consensus of the scientific community is that it is linear. And if those people who argue that it is not, they still haven't produced any evidence to support that notion, as far as I am concerned.

Q Do you know what the U.S. EPA's position is on whether there is a linear or nonlinear dose response curve for dioxin in human health effects?

A It is my understanding that they believe it is linear.

Q Do you know what the reason -- do you know the reason the U.S. EPA gives for its approach?

A Well, that is general policy. And as I say, most scientists who are in the field feel that there is no threshold for cancer induction, basically, for the reasons that we have talked about.

Namely, that if the chemical is capable of having an adverse effect, it is very difficult to imagine how it would have a threshold.

Q So for the EPA, at least, the use of a nonlinear dose -- the use of a linear dose response curve reflects policy decision as opposed to a decision based on the weight of the evidence; correct?

A No, I think that it is based on the weight of the evidence. At this point, there is no scientific evidence that there is a threshold for cancer induction.

There are people that argue it theoretically, but they have presented no data to support it.

MR. HOPP: Now is a convenient time, if you want to stop.

MR. PRUDHOMME: Okay.

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I, JAMES DAHLGREN, M.D., do hereby declare under penalty of perjury that I have read the foregoing transcript; that I have made any corrections as appear noted, in ink, initialed by me, or attached hereto; that my testimony as contained herein, as corrected, is true and correct.

EXECUTED this _____ day of _____,

20__, at _____, _____.

(City) (State)

_____

JAMES DAHLGREN, M.D.

I, the undersigned, a Certified Shorthand Reporter of the State of California, do hereby certify:

That the foregoing proceedings were taken before me at the time and place herein set forth; that any witnesses in the foregoing proceedings, prior to testifying, were placed under oath; that a verbatim record of the proceedings was made by me using machine shorthand which was thereafter transcribed under my direction; further, that the foregoing is an accurate transcription thereof.

I further certify that I am neither financially interested in the action nor a relative or employee of any attorney of any of the parties.

IN WITNESS WHEREOF, I have this date subscribed my name.

Dated: _____

_____

Diana Janniere

CSR No. 10034